* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	OCT 04	Precision of EMBASE searching enhanced with new chemical name field
NEWS 3	OCT 06	Increase your retrieval consistency with new formats or for Taiwanese application numbers in CA/CAplus.
NEWS 4	OCT 21	CA/CAplus kind code changes for Chinese patents increase consistency, save time
NEWS 5	OCT 22	New version of STN Viewer preserves custom highlighting of terms when patent documents are saved in .rtf format
NEWS 6	OCT 28	INPADOCDB/INPAFAMDB: Enhancements to the US national patent classification.
NEWS 7	NOV 03	New format for Korean patent application numbers in CA/CAplus increases consistency, saves time.
NEWS 8	NOV 04	Selected STN databases scheduled for removal on December 31, 2010
NEWS 9	NOV 18	PROUSDDR and SYNTHLINE Scheduled for Removal December 31, 2010 by Request of Prous Science
NEWS 10	NOV 22	Higher System Limits Increase the Power of STN Substance-Based Searching
NEWS 11	NOV 24	Search an additional 46,850 records with MEDLINE backfile extension to 1946
NEWS 12	DEC 14	New PNK Field Allows More Precise Crossover among STN Patent Databases
NEWS 13	DEC 18	ReaxysFile available on STN
NEWS 14	DEC 21	CAS Learning Solutions -- a new online training experience
NEWS 15	DEC 22	Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAplus
NEWS 16	JAN 24	The new and enhanced DPCI file on STN has been released
NEWS 17	JAN 26	Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents
NEWS 18	JAN 26	Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE
NEWS 19	JAN 28	CABA will be updated weekly
NEWS 20	FEB 23	PCTFULL file on STN completely reloaded
NEWS 21	FEB 23	STN AnaVist Test Projects Now Available for Qualified Customers
NEWS 22	FEB 25	LPCI will be replaced by LDPCI
NEWS 23	MAR 07	Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN

NEWS EXPRESS 17 DECEMBER 2010 CURRENT WINDOWS VERSION IS V8.4.2 .1,
AND CURRENT DISCOVER FILE IS DATED 24 JANUARY 2011.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items

Enter NEWS followed by the item number or name to see news on that specific topic.

```
* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 14:00:49 ON 06 APR 2011

=> file caplus
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.23       0.23

FILE 'CAPLUS' ENTERED AT 14:00:57 ON 06 APR 2011
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2011 AMERICAN CHEMICAL SOCIETY (ACS)


Copyright of the articles to which records in this database refer is
held by the publishers listed in the PUBLISHER (PB) field (available
for records published or updated in Chemical Abstracts after December
26, 1996), unless otherwise indicated in the original publications.
The CA Lexicon is the copyrighted intellectual property of the
American Chemical Society and is provided to assist you in searching
databases on STN.  Any dissemination, distribution, copying, or storing
of this information, without the prior written consent of CAS, is
strictly prohibited.

FILE COVERS 1907 - 6 Apr 2011  VOL 154 ISS 15
FILE LAST UPDATED: 5 Apr 2011  (20110405/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Feb 2011
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Feb 2011

CAplus now includes complete International Patent Classification (IPC)
reclassification data for the fourth quarter of 2010.

CAS Information Use Policies apply and are available at:

http://www.cas.org/legal/infopolicy.html

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> OX40 not OX40L
           733 OX40
           338 OX40L
L1         490 OX40 NOT OX40L

=> adjuvant (s) L1
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'ADJUVANT (S) L1'
         55120 ADJUVANT
         31605 ADJUVANTS
         71536 ADJUVANT
                  (ADJUVANT OR ADJUVANTS)
L2          32 ADJUVANT (S) L1

=> D L2 IBIB ABS 1-32

L2   ANSWER 1 OF 32  CAPLUS  COPYRIGHT 2011 ACS on STN
```

Full Text | Find References

ACCESSION NUMBER: 2011:359257 CAPLUS
TITLE: The function of follicular helper T cells is regulated by the strength of T cell antigen receptor binding. [Erratum to document cited in CA150:281269]
AUTHOR(S): Fazilleau, Nicolas; McHeyzer-Williams, Louise J.; Rosen, Hugh; McHeyzer-Williams, Michael G.
CORPORATE SOURCE: Department of Immunology and Microbial Sciences, The Scripps Research Institute, La Jolla, CA, USA
SOURCE: Nature Immunology (2011), 12(4), 362-363
CODEN: NIAMCZ; ISSN: 1529-2908
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; Errata; (online computer file)
LANGUAGE: English
AB This article was published online 1 March 2009; cor. online 8 March 2009; addendum published after print 8 March 2011.

L2 ANSWER 2 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Cited References

ACCESSION NUMBER: 2011:183680 CAPLUS
TITLE: Anti-GITR antibodies - potential clinical applications for tumor immunotherapy
AUTHOR(S): Schaer, David A.; Cohen, Adam D.; Wolchok, Jedd D.
CORPORATE SOURCE: Immunology Program, Sloan-Kettering Institute for Cancer Research, New York, NY, 10065, USA
SOURCE: Current Opinion in Investigational Drugs (BioMed Central) (2010), 11(12), 1378-1386
CODEN: COIDAZ; ISSN: 2040-3429
URL: http://www.biomedcentral.com/content/pdf/cd-1152427.pdf
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Since the development of the first vaccines, modern medicine has been consistently aiming to improve the efficacy of immune responses. Traditionally, **adjuvants** have been used as non-specific immune modulators to enhance recognition and activation against a desired antigen. By providing 'danger' signals to the immune system, **adjuvants** activate innate immunity, which enhances the development of protective and therapeutic adaptive immune responses. The newest class of immune modulators bypasses the innate response and targets cells of the adoptive response directly. Targeted immunomodulatory therapy is focused primarily on the activation of costimulatory receptors (eg, 4-1BB, **OX40** and GITR [glucocorticoid-induced TNF receptor-related gene]) or the blockade of co-inhibitory receptors (eg, CTLA-4, PD-1 and PD-L1) on T-cells during activation and/or effector responses. With promising clin. results obtained to date, immunomodulatory therapy is becoming an integral part of immunotherapeutic approaches. The modulation of GITR is listed as one of the top 25 most promising research areas by the NCI, and has demonstrated potential in both antitumor and vaccine settings. This review discusses the role of GITR as a potential target for immunomodulatory therapy, as well as the research involved in understanding the mechanisms of anti-GITR therapy and current progress in translation into the clinic.
REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Cited References

ACCESSION NUMBER: 2011:54732 CAPLUS

DOCUMENT NUMBER: 154:200189
TITLE: Preparation of human papillomavirus containing mutant E6 and E7 antigens and its use as immunostimulant for preventing or treating cervical cancer
INVENTOR(S): Sung, Yeong Cheol; Seo, Sang Hwan; Seo, Yu Seok
PATENT ASSIGNEE(S): Genexine, Inc., S. Korea; Biod Co., Ltd.
SOURCE: Repub. Korean Kongkae Taeho Kongbo, 20pp.
CODEN: KRXXA7
DOCUMENT TYPE: Patent
LANGUAGE: Korean
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
KR 2011002730	A	20110110	KR 2009-60348	20090702

PRIORITY APPLN. INFO.: KR 2009-60348 20090702

AB This invention provides a process of prepn. of human papillomavirus contg. mutant E6 and E7 antigens. The three-dimensional structure of E6 and E7 antigens of HPV type 16 and HPV type 18 was modified by mutagenesis. The DNA and protein sequences of E6 and E7 fusion antigen, signal peptide and immune **adjuvant** peptides were disclosed. The human papillomavirus contg. E6 and E7 fusion antigen can be used as immunostimulant for preventing or treating cervical cancer.

L2 ANSWER 4 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2010:1501342 CAPLUS
DOCUMENT NUMBER: 154:1859
TITLE: Recombinant multiple domain fusion protein mitogens and use thereof for inducing enhancement or repression of antigen-specific immunity
INVENTOR(S): Ochi, Atsuo
PATENT ASSIGNEE(S): Can.
SOURCE: U.S. Pat. Appl. Publ., 114pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20100303811	A1	20101202	US 2009-483876	20090612

PRIORITY APPLN. INFO.: US 2008-73010P P 20080616

AB The invention relates to cell stimulatory fusion proteins and DNA sequences, vectors comprising at least two agonists of TNF/TNFR superfamily, Ig superfamily, cytokine family proteins, and optional antigen combinations. Instructions for use of these proteins and DNA constructs as immune **adjuvants** and vaccines for treatment of various chronic diseases such as viral infection are also provided. Addnl., the use of these protein and DNA constructs as immune suppressants for treatment of various chronic diseases, such as autoimmunity and organ transplant rejection, is also illustrated. Particularly, this invention provides nucleic acid constructs contg. genes encoding sol. fusion proteins which comprise: (i) a CD40 ligand, a Fas ligand extracellular domain, and an IgG Fc domain; (ii) a CD28 ligand (B7-2), a Fas ligand extracellular domain, and an IgG Fc domain; (iii) an **OX40** ligand, a 4-1BB ligand extracellular domain, and an IgG Fc domain; (iv) a CD40

ligand, a ICOS extracellular domain, and an IgG Fc domain; (v) a NGFβ ligand, a Fas ligand extracellular domain, and an IgG Fc domain; (vi) an interleukin-2 ligand, a Fas ligand extracellular domain, and an IgG Fc domain. The fusion proteins will preferably elicit a de novo effect to cause immune cell activation relative to when any of the resp. agonistic polypeptides contained therein are administered alone.

L2 ANSWER 5 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Info. References

ACCESSION NUMBER: 2010:1210899 CAPLUS
TITLE: **Adjuvant** therapy with agonistic antibodies to CD134 (**OX40**) increases local control after surgical or radiation therapy of cancer in mice
AUTHOR(S): Gough, Michael J.; Crittenden, Marka R.; Sarff, MaryClare; Pang, Puiyi; Seung, Steven K.; Vetto, John T.; Hu, Hong-Ming; Redmond, William L.; Holland, John; Weinberg, Andrew D.
CORPORATE SOURCE: Earle A. Chiles Research Institute, Robert W. Franz Cancer Center, Providence Cancer Center The Oregon Clinic, Portland, OR, USA
SOURCE: Journal of Immunotherapy (2010), 33(8), 798-809 CODEN: JOIMF8; ISSN: 1524-9557
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The tumor recurrence from residual local or micrometastatic disease remains a problem in cancer therapy. In patients with soft tissue sarcoma and the patients with inoperable nonsmall cell lung cancer, local recurrence is common and significant mortality is caused by the subsequent emergence of metastatic disease. Thus, although the aim of the primary therapy is curative, the outcome may be improved by addnl. targeting of residual microscopic disease. We display in a murine model that surgical removal of a large primary sarcoma results in local recurrence in approx. 50% of animals. Depletion of CD8 T cells results in local recurrence in 100% of animals, indicating that these cells are involved in the control of residual disease. We further show that systemic **adjuvant** administration of α**OX40** at surgery eliminates local recurrences. In this model, α**OX40** acts to directly enhance tumor antigen-specific CD8 T-cell proliferation in the lymph node draining the surgical site, and results in increased tumor antigen-specific cytotoxicity in vivo. These results are also corroborated in a murine model of hypofractionated radiation therapy of lung cancer. Administration of α**OX40** in combination with radiation significantly extended the survival compared with either agent alone, and resulted in a significant proportion of long-term tumor-free survivors. We conclude that α**OX40** increases tumor antigen-specific CD8 T-cell cytotoxic activity resulting in improved endogenous immune control of residual microscopic disease, and we propose that **adjuvant** α**OX40** administration may be a valuable addn. to surgical and radiation therapy for cancer.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2010:335925 CAPLUS

DOCUMENT NUMBER: 153:283633
TITLE: Costimulation signals for memory CD8+ T cells during viral infections
AUTHOR(S): Duttagupta, Priyanka A.; Boesteanu, Alina C.; Katsikis, Peter D.
CORPORATE SOURCE: Department of Microbiology and Immunology and Center for Immunology and Vaccine Science, Drexel University College of Medicine, Philadelphia, PA, USA
SOURCE: Critical Reviews in Immunology (2009), 29(6), 469-486
CODEN: CCRIDE; ISSN: 1040-8401
PUBLISHER: Begell House, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Costimulation signals have been recognized as crit. for optimal T-cell responses and result from important interactions between receptors on the surface of T cells and their ligands on antigen-presenting cells. Two families of receptors, the CD28 family and the tumor necrosis factor receptor (TNFR) family, have been found to be major players in providing costimulation to CD8+ T cells. Recent studies using viral infection models have highlighted the importance of CD28 costimulation signals during memory responses against viruses. Programmed death-1 (PD-1), another member of the CD28 family, may contribute to functional defects of helpless memory CD8+ T cells. Members of the TNFR family, such as CD27, 4-1BB, CD40, TRAIL (tumor necrosis factor-related apoptosis-inducing ligand), and **OX40**, have also been implicated in the survival, generation, maintenance, and quality of virus-specific memory CD8+ T cells. The delivery of costimulatory mols. such as CD28, 4-1BB, and **OX40** can help boost the generation and function of virus-specific memory CD8+ T cells. The use of costimulatory mols. as **adjuvants**, along with viral antigens in vaccines, may facilitate the generation of effective antigen-specific memory CD8+ T-cell responses. Understanding the costimulatory requirements of memory CD8+ T cells, therefore, may lead to improved vaccines that target anti-viral CD8+ T-cell memory.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 138 THERE ARE 138 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Cited References

ACCESSION NUMBER: 2009:1588570 CAPLUS
DOCUMENT NUMBER: 152:136176
TITLE: Methylprednisolone inhibits IFN-γ and IL-17 expression and production by cells infiltrating central nervous system in experimental autoimmune encephalomyelitis
AUTHOR(S): Miljkovic, Zeljka; Momcilovic, Miljana; Miljkovic, Djordje; Mostarica-Stojkovic, Marija
CORPORATE SOURCE: Institute of Microbiology and Immunology, School of Medicine, University of Belgrade, Belgrade, Serbia
SOURCE: Journal of Neuroinflammation (2009), 6, No pp. given
CODEN: JNOEB3; ISSN: 1742-2094
URL: http://www.jneuroinflammation.com/content/pdf/1742-2094-6-37.pdf
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Background: Glucocorticoids have been shown to be effective in the

treatment of autoimmune diseases of the CNS such as multiple sclerosis and its animal model, exptl. autoimmune encephalomyelitis (EAE). However, the mechanisms and the site of glucocorticoids' actions are still not completely defined. The aim of this study was to investigate the in vivo effect of the synthetic glucocorticoid methylprednisolone (MP) on the expression and prodn. of proinflammatory cytokines interferon (IFN)-γ and interleukin (IL)-17 by cells infiltrating CNS tissue. Methods: Exptl. autoimmune encephalomyelitis was induced in Dark Agouti (DA) rats by immunization with rat spinal cord homogenate mixed with **adjuvants**. Commencing on the day when the first EAE signs appeared, DA rats were injected daily for 3 days with MP and/or RU486, an antagonist of glucocorticoid receptor. Cytokine prodn. and gene expression in CNS-infiltrating cells and lymph node cells were measured using ELISA and real time PCR, resp. Results: Treatment of rats with MP ameliorated EAE, and the animals recovered without relapses. Further, MP inhibited IFN-γ and IL-17 expression and prodn. in cells isolated from the CNS of DA rats with EAE after the last injection of MP. The obsd. effect of MP in vivo treatment was not mediated through depletion of CD4+ T cells among CNS infiltrating cells, or through induction of their apoptosis within the CNS. Finally, the glucocorticoid receptor-antagonist RU486 prevented the inhibitory effect of MP on IFN-γ and IL-17 prodn. both in vitro and in vivo, thus indicating that the obsd. effects of MP were mediated through glucocorticoid receptor-dependent mechanisms. Conclusion: Taken together, these results demonstrate that amelioration of EAE by exogenous glucocorticoids might be, at least partly, ascribed to the limitation of effector cell functions in the target tissue.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | CAPLUS References

ACCESSION NUMBER: 2009:1556173 CAPLUS
DOCUMENT NUMBER: 153:141678
TITLE: Timing and tunning of CD27-CD70 interactions: the impact of signal strength in setting the balance between adaptive responses and immunopathology
AUTHOR(S): Nolte, Martijn A.; van Olffen, Ronald W.; van Gisbergen, Klaas P. J. M.; van Lier, Rene A. W.
CORPORATE SOURCE: Department of Experimental Immunology, Academic Medical Center, University of Amsterdam, Amsterdam, Neth.
SOURCE: Immunological Reviews (2009), 229(1), 216-231
CODEN: IMRED2; ISSN: 1600-065X
URL: http://www3.interscience.wiley.com/cgi-bin/fulltext/122341683/PDFSTART
PUBLISHER: John Wiley & Sons, Inc.
DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English

AB A review. After binding its natural ligand cluster of differentiation 70 (CD70), CD27, a tumor necrosis factor receptor (TNFR)-assocd. factor-binding member of the TNFR family, regulates cellular activity in subsets of T, B, and natural killer cells as well as hematopoietic progenitor cells. In normal immune responses, CD27 signaling appears to be limited predominantly by the restricted expression of CD70, which is only transiently expressed by cells of the immune system upon activation. Studies performed in CD27-deficient and CD70-transgenic mice have defined a non-redundant role of this receptor-ligand pair in shaping adaptive T-cell responses. Moreover, **adjuvant** properties of CD70 have been exploited for the design of anti-cancer vaccines. However, continuous

CD27-CD70 interactions may cause immune dysregulation and immunopathol. in conditions of chronic immune activation such as during persistent virus infection and autoimmune disease. We conclude that optimal tuning of CD27-CD70 interaction is crucial for the regulation of the cellular immune response. We provide a detailed comparison of costimulation through CD27 with its closely related family members 4-1BB (GD137), CD30, herpes virus entry mediator, **OX40** (CD134), and glucocorticoid-induced TNFR family-related gene, and we argue that these receptors do not have a unique function per se but that rather the timing, context, and intensity of these costimulatory signals det. the functional consequence of their activity.

OS.CITING REF COUNT: 17 THERE ARE 17 CAPLUS RECORDS THAT CITE THIS RECORD (17 CITINGS)

REFERENCE COUNT: 144 THERE ARE 144 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2009:510941 CAPLUS
DOCUMENT NUMBER: 151:484817
TITLE: The anti-cancer agents lenalidomide and pomalidomide inhibit the proliferation and function of T regulatory cells
AUTHOR(S): Galustian, Christine; Meyer, Brendan; Labarthe, Marie-Christine; Dredge, Keith; Klaschka, Deborah; Henry, Jake; Todryk, Stephen; Chen, Roger; Muller, George; Stirling, David; Schafer, Peter; Bartlett, J. Blake; Dalgleish, Angus G.
CORPORATE SOURCE: Department of Oncology, St Georges University of London, London, UK
SOURCE: Cancer Immunology Immunotherapy (2009), 58(7), 1033-1045
CODEN: CIIMDN; ISSN: 0340-7004
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Lenalidomide (Revlimid; CC-5013) and pomalidomide (CC-4047) are IMiDs proprietary drugs having immunomodulatory properties that have both shown activity in cancer clin. trials; lenalidomide is approved in the United States for a subset of MDS patients and for treatment of patients with multiple myeloma when used in combination with dexamethasone. These drugs exhibit a range of interesting clin. properties, including anti-angiogenic, anti-proliferative, and pro-erythropoietic activities although exact cellular target(s) remain unclear. Also, anti-inflammatory effects on LPS-stimulated monocytes (TNF-α is decreased) and costimulatory effects on anti-CD3 stimulated T cells, (enhanced T cell proliferation and proinflammatory cytokine prodn.) are obsd. These drugs also cause augmentation of NK-cell cytotoxic activity against tumor-cell targets. Having shown that pomalidomide confers T cell-dependant **adjuvant**-like protection in a preclin. whole tumor-cell vaccine-model, we now show that lenalidomide and pomalidomide strongly inhibit T-regulatory cell proliferation and suppressor-function. Both drugs inhibit IL-2-mediated generation of FOXP3 pos. CTLA-4 pos. CD25high CD4+ T regulatory cells from PBMCs by up to 50%. Furthermore, suppressor function of pre-treated T regulatory cells against autologous responder-cells is abolished or markedly inhibited without drug related cytotoxicity. Also, Balb/C mice exhibit 25% redn. of lymph-node T regulatory cells after pomalidomide treatment. Inhibition of T regulatory

cell function was not due to changes in TGF-β or IL-10 prodn. but was assocd. with decreased T regulatory cell FOXP3 expression. In conclusion, our data provide one explanation for **adjuvant** properties of lenalidomide and pomalidomide and suggest that they may help overcome an important barrier to tumor-specific immunity in cancer patients.

OS.CITING REF COUNT: 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (22 CITINGS)

REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 10 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:247868 CAPLUS
DOCUMENT NUMBER: 150:281269
TITLE: The function of follicular helper T cells is regulated by the strength of T cell antigen receptor binding
AUTHOR(S): Fazilleau, Nicolas; McHeyzer-Williams, Louise J.; Rosen, Hugh; McHeyzer-Williams, Michael G.
CORPORATE SOURCE: Department of Immunology and Microbial Sciences, The Scripps Research Institute, La Jolla, CA, USA
SOURCE: Nature Immunology (2009), 10(4), 375-384
CODEN: NIAMCZ; ISSN: 1529-2908
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB How follicular helper T cells (TFH cells) differentiate to regulate B cell immunity is crit. for effective protein vaccination. Here we define three transcription factor T-bet-expressing antigen-specific effector helper T cell subsets with distinguishable function, migratory properties and developmental programming in vivo. Expression of the transcriptional repressor Blimp-1 distinguished T zone 'lymphoid' effector helper T cells (CD62LhiCCR7hi) from CXCR5lo 'emigrant' effector helper T cells and CXCR5hi 'resident' TFH cells expressing the transcriptional repressor Bcl-6 (CD62LloCCR7lo). We then show by adoptive transfer and intact polyclonal responses that helper T cells with the highest specific binding of peptide-major histocompatibility complex class II and the most restricted T cell antigen receptor junctional diversity 'preferentially' developed into the antigen-specific effector TFH compartment. Our studies demonstrate a central function for differences in the binding strength of the T cell antigen receptor in the antigen-specific mechanisms that 'program' specialized effector TFH function in vivo.

OS.CITING REF COUNT: 45 THERE ARE 45 CAPLUS RECORDS THAT CITE THIS RECORD (45 CITINGS)

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2009:156836 CAPLUS
DOCUMENT NUMBER: 150:189261
TITLE: Asthma-Related Environmental Fungus, Alternaria, Activates Dendritic Cells and Produces Potent Th2 **Adjuvant** Activity
AUTHOR(S): Kobayashi, Takao; Iijima, Koji; Radhakrishnan, Suresh; Mehta, Vinay; Vassallo, Robert; Lawrence, Christopher B.; Cyong, Jong-Chol; Pease, Larry R.; Oguchi, Katsuji; Kita, Hirohito
CORPORATE SOURCE: Division of Allergic Diseases, Department of Internal

Medicine, Mayo Clinic, Rochester, MN, 55905, USA
SOURCE: Journal of Immunology (2009), 182(4), 2502-2510
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Asthma is thought to result from dysregulated Th2-like airway inflammatory responses to the environment. Although the etiol. of asthma is not fully understood in humans, clin. and epidemiol. evidence suggest a potential link between exposure to environmental fungi, such as Alternaria, and development and/or exacerbation of asthma. The goal of this project was to investigate the mechanisms of airway Th2 responses by using Alternaria as a clin. relevant model for environmental exposure. Airway exposure of naive animals to an exptl. Ag, OVA, or a common allergen, short ragweed pollen, induced no or minimal immune responses to these Ags. In contrast, mice developed strong Th2-like immune responses when they were exposed to these Ags in the presence of Alternaria ext. Exts. of other fungi, such as Aspergillus and Candida, showed similar Th2 **adjuvant** effects, albeit not as potently. Alternaria stimulated bone marrow-derived dendritic cells (DCs) to express MHC class II and costimulatory mols., including **OX40** ligand, in vitro. Importantly, Alternaria inhibited IL-12 prodn. by activated DCs, and DCs exposed to Alternaria enhanced Th2 polarization of CD4+ T cells. Furthermore, adoptive airway transfer of DCs, which had been pulsed with OVA in the presence of Alternaria, showed that the recipient mice had enhanced IgE Ab prodn. and Th2-like airway responses to OVA. Thus, the asthma-related environmental fungus Alternaria produces potent Th2-like **adjuvant** effects in the airways. Such immunogenic properties of certain environmental fungi may explain their strong relationships with human asthma and allergic diseases.
OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 12 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | 52 References
ACCESSION NUMBER: 2008:1204405 CAPLUS
DOCUMENT NUMBER: 149:400022
TITLE: Optimising anti-tumour CD8 T-cell responses using combinations of immunomodulatory antibodies
AUTHOR(S): Gray, Juliet C.; French, Ruth R.; James, Sonya; Al-Shamkhani, Aymen; Johnson, Peter W.; Glennie, Martin J.
CORPORATE SOURCE: Tenovus Research Laboratory, Cancer Sciences Division, Southampton University School of Medicine, Southampton, UK
SOURCE: European Journal of Immunology (2008), 38(9), 2499-2511
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH & Co. KGaA
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Immunostimulatory mAb as vaccine **adjuvants** for the treatment of cancer hold considerable potential for boosting weak responses when used against immunogenic tumors, or in combination with various other vaccines. We now show that when administered with OVA, the combination of anti-4-1BB mAb with anti-CD40, anti-**OX40** or anti-CD25 resulted in a fourfold enhancement in the antigen-specific T-cell response compared with anti-4-1BB mAb alone, with a similar enhancement in memory responses

following rechallenge with OVA. Although the no. of antigen-specific T-cells generated after treatment with each of the combinations was similar, marked functional differences were detected. In particular, anti-4-1BB/anti-CD25 resulted in excellent expansion of specific CD8+ T cells but produced fewer IFN-γ-secreting effector cells than the other combinations. Anti-4-1BB/anti-**OX40** proved to be the most potent, inducing the most effective T-cell responses in the RIPmOVA diabetes model with adoptively transferred OVA-specific T cells, and, when given with a peptide vaccine, protecting mice against the poorly immunogenic B16-F10 tumor. Overall the results suggest that although these combinations of mAb look promising in terms of their therapeutic potential, further functional assays are needed to compare their effects.

OS.CITING REF COUNT: 11 THERE ARE 11 CAPLUS RECORDS THAT CITE THIS RECORD (11 CITINGS)

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 13 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2008:1192185 CAPLUS

DOCUMENT NUMBER: 149:462474

TITLE: Imidazoquinoline Acts as Immune **Adjuvant** for Functional Alteration of Thymic Stromal Lymphopoietin-Mediated Allergic T Cell Response

AUTHOR(S): Torii, Yoshitaro; Ito, Tomoki; Amakawa, Ryuichi; Sugimoto, Hiroyuki; Amuro, Hideki; Tanijiri, Tsutomu; Katashiba, Yuichi; Ogata, Makoto; Yokoi, Takashi; Fukuhara, Shirou

CORPORATE SOURCE: First Department of Internal Medicine, Kansai Medical University, Osaka, 570-8506, Japan

SOURCE: Journal of Immunology (2008), 181(8), 5340-5349 CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Atopic dermatitis is a major allergic disease that develops through dysregulation of Th2-mediated inflammation. Although dendritic cells (DCs) have been thought to play a crit. role in the upstream phase of the allergic cascade, conventional drugs such as steroids and chem. mediator antagonists target the effector cells or factors in allergic inflammation. Recently, it has been demonstrated that interaction between thymic stromal lymphopoietin (TSLP) and human DCs plays an essential role in evoking inflammatory Th2 responses in allergy through **OX40** ligand expression on DCs. In this study, we provide evidence that R848, an imidazoquinoline compd., which is a TLR ligand and a strong Th1 response-inducing reagent, is a potent **adjuvant** for the alteration of the Th2-inducing potency of human DCs activated by TSLP (TSLP-DCs). R848 inhibited the inflammatory Th2-inducing capacity of TSLP-DCs and redirected them to possessing an IL-10 and IFN-γ-producing regulatory Th1-inducing capacity. This functional alteration depended on both repression of **OX40** ligand expression and induction of IL-12 prodn. from DCs by the addn. of R848. Addnl., R848 had the ability to inhibit the TSLP-mediated expansion and maintenance of the Th2 memory response. These findings suggest that imidazoquinoline may be a useful in the treatment of allergic diseases that are triggered by TSLP.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 14 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:1141695 CAPLUS
DOCUMENT NUMBER: 149:491968
TITLE: Mycobacterium bovis Bacillus Calmette-Guerin suppresses inflammatory Th2 responses by inducing functional alteration of TSLP-activated dendritic cells
AUTHOR(S): Yokoi, Takashi; Amakawa, Ryuichi; Tanijiri, Tsutomu; Sugimoto, Hiroyuki; Torii, Yoshitaro; Amuro, Hideki; Son, Yonsu; Tajima, Kenichirou; Liu, Yong-Jun; Ito, Tomoki; Fukuhara, Shirou
CORPORATE SOURCE: First Department of Internal Medicine, Kansai Medical University, Osaka, Japan
SOURCE: International Immunology (2008), 20(10), 1321-1329
CODEN: INIMEN; ISSN: 0953-8178
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Allergic diseases such as atopic dermatitis and asthma develop as a consequence of dysregulated Th2 responses. Recently, it has been demonstrated that interaction between dendritic cells (DCs) and thymic stromal lymphopoietin (TSLP), an IL-7-like cytokine, is essential for evoking Th2 responses in allergy. In this study, we investigated whether Mycobacterium bovis Bacillus Calmette-Guerin (BCG), a strong Th1 response-inducing **adjuvant**, can alter the function of DCs activated by TSLP (TSLP-DCs). We demonstrated that BCG redirects TSLP-DCs away from inducing inflammatory Th2 cells that produce IL-4, IL-5, IL-13 and tumor necrosis factor (TNF)-α and toward regulatory Th1 cells that produce IFN-γ and IL-10. We also demonstrated that this functional alteration of TSLP-DCs by BCG depended on both prodn. of IL-12 from DCs and down-regulation of **OX40** ligand, a member of the TNF family, on DCs. These findings suggest that BCG might be a useful **adjuvant** for the treatment of allergic diseases that are triggered by TSLP.
OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 15 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:381634 CAPLUS
DOCUMENT NUMBER: 149:51274
TITLE: **Adjuvant** effect of anti-4-1BB mAb administration in adoptive T cell therapy of cancer
AUTHOR(S): Li, Qiao; Iuchi, Takekazu; Jure-Kunkel, Maria N.; Chang, Alfred E.
CORPORATE SOURCE: Division of Surgical Oncology, University of Michigan, Ann Arbor, MI, 48109-5932, USA
SOURCE: International Journal of Biological Sciences (2007), 3(7), 455-462
CODEN: IJBSB9; ISSN: 1449-2288
URL: http://www.biolsci.org/v03p0455.pdf
PUBLISHER: Ivyspring International Publisher
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English
AB Administration of anti-4-1BB mAb has been found to be a potent **adjuvant**

when combined with other therapeutic approaches, e.g. chemotherapy, cytokine therapies, anti-**OX40** therapy, and peptide or DC vaccines. However, the **adjuvant** effect of anti-4-1BB mAb administration in adoptive T cell therapy of cancer has not been fully evaluated. In this report, effector T cells were generated in vitro by anti-CD3/anti-CD28 activation of tumor-draining lymph node (TDLN) cells and used in an adoptive immunotherapy model. While T cells or anti-4-1BB alone showed no therapeutic efficacy in mice bearing macroscopic 10-day pulmonary metastases, T cells plus anti-4-1BB mediated significant tumor regression in an anti-4-1BB dose dependent manner. Mice bearing microscopic 3-day lung metastases treated with T cells alone demonstrated tumor regression which was significantly enhanced by anti-4-1BB administration. NK cell depletion abrogated the augmented therapeutic efficacy rendered by anti-4-1BB. Cell transfer between congenic hosts demonstrated that anti-4-1BB administration increased the survival of adoptively transferred TDLN cells. Using STAT4-/- mice, we found that modulated IFNγ secretion in wt TDLN cells after anti-CD3/CD28/4-1BB activation in vitro was lost in similarly stimulated STAT4-/- TDLN cells. Addnl., anti-4-1BB administration failed to augment the therapeutic efficacy of T cell therapy in STAT4-/- mice. Together, these results indicate that administered anti-4-1BB mAb can serve as an effective **adjuvant** to augment the antitumor reactivity of adoptively transferred T cells by recruiting the host NK cells; increasing the persistence of infused effector T cells, and modulating the STAT4 mol. signaling pathway.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 16 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2008:46087 CAPLUS
DOCUMENT NUMBER: 148:119833
TITLE: Identification and monitoring of effector and regulatory T cells during experimental arthritis based on differential expression of CD25 and CD134
AUTHOR(S): Nolte-'t Hoen, Esther N. M.; Boot, Elmieke P. J.; Wagenaar-Hilbers, Josee P. A.; van Bilsen, Jolanda H. M.; Arkesteijn, Ger J. A.; Storm, Gert; Everse, Linda A.; van Eden, Willem; Wauben, Marca H. M.
CORPORATE SOURCE: Departments of Biochemistry and Cell Biology, Utrecht University, Utrecht, Neth.
SOURCE: Journal of Leukocyte Biology (2008), 83(1), 112-121 CODEN: JLBIE7; ISSN: 0741-5400
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Major problems in the anal. of CD4+ effector cell and regulatory T cell (Treg) populations in an activated immune system are caused by the facts that both cell types can express CD25 and that the discriminatory marker forkhead box p3 can only be analyzed in nonviable (permeabilized) cells. Here, we show that CD134 (**OX40**) can be used as a discriminatory marker combined with CD25 to isolate and characterize viable CD4+ effector cells and Tregs. Before and during **adjuvant** arthritis in rats, coexpression of CD134 and CD25 identified activated Tregs consistently, as these T cells proliferated poorly to disease-assocd. antigens and were suppressive in vitro and in vivo. Depending on the time of isolation and location, CD4+ T cell populations expressing CD134 or CD25 contained effector/memory

T cells. Anal. of the function, phenotype, and amt. of the CD4+ T cell subsets in different lymph node stations revealed spatiotemporal differences in effector cell and Treg compartments during exptl. arthritis.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 17 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:1248032 CAPLUS
DOCUMENT NUMBER: 148:9283
TITLE: The Lipopolysaccharide **Adjuvant** Effect on T Cells Relies on Nonoverlapping Contributions from the MyD88 Pathway and CD11c+ Cells
AUTHOR(S): McAleer, Jeremy P.; Zammit, David J.; Lefrancois, Leo; Rossi, Robert J.; Vella, Anthony T.
CORPORATE SOURCE: Department of Immunology, University of Connecticut Health Center, Farmington, CT, 06030, USA
SOURCE: Journal of Immunology (2007), 179(10), 6524-6535 CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Bacterial LPS is a natural **adjuvant** that induces profound effects on T cell clonal expansion, effector differentiation, and long-term T cell survival. Here, the authors delineate the in vivo mechanism of LPS action by pinpointing a role for MyD88 and CD11c+ cells. LPS induced long-term survival of superantigen-stimulated CD4 and CD8 T cells in a MyD88-dependent manner. By tracing peptide-stimulated CD4 T cells after adoptive transfer, the authors showed that for LPS to mediate T cell survival, the recipient mice were required to express MyD88. Even when peptide-specific CD4 T cell clonal expansion was dramatically boosted by enforced **OX40** costimulation, **OX40** only synergized with LPS to induce survival when the recipient mice expressed MyD88. Nevertheless, these activated, but moribund, T cells in the MyD88-/- mice acquired effector properties, such as the ability to synthesize IFN-γ, demonstrating that effector differentiation is not automatically coupled to a survival program. The authors confirmed this notion in reverse fashion by showing that effector differentiation was not required for the induction of T cell survival. Hence, depletion of CD11c+ cells did not affect LPS-driven specific T cell survival, but CD11c+ cells were paramount for optimal effector T cell differentiation as measured by IFN-γ potential. Thus, LPS adjuvanticity is based on MyD88 promoting T cell survival, while CD11c+ cells support effector T cell differentiation.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 18 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:1206472 CAPLUS
DOCUMENT NUMBER: 145:504036
TITLE: Trimeric OX-40-immunoglobulin fusion protein as **adjuvant** for enhancing antigen-specific immune responses

INVENTOR(S): Weinberg, Andrew D.; Morris, Nicholas P.; Peters, Carmen
PATENT ASSIGNEE(S): Providence Health System, USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006121810	A2	20061116	WO 2006-US17285	20060504
WO 2006121810	A3	20070329		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2006244497	A1	20061116	AU 2006-244497	20060504
CA 2606809	A1	20061116	CA 2006-2606809	20060504
US 20060280728	A1	20061214	US 2006-418940	20060504
EP 1877090	A2	20080116	EP 2006-770019	20060504
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
US 20100136032	A1	20100603	US 2009-618678	20091113

PRIORITY APPLN. INFO.:

US 2005-678420P	P	20050506
US 2006-418940	B1	20060504
WO 2006-US17285	W	20060504

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Compns. including a trimeric OX-40 fusion protein are disclosed. The trimeric OX-40 fusion protein comprises an Ig Fc domain, a trimerization isoleucine zipper domain and a OX-40 receptor binding domain. Also disclosed are methods for enhancing the immune response of a mammal to an antigen by engaging the OX-40 receptor on the surface of T-cells involving administering to the mammal a compn. comprising a trimeric OX-40 fusion protein and a pharmaceutically acceptable carrier. The antigen is a bacterial antigen, viral antigen or tumor antigen.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 19 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2006:191503 CAPLUS
DOCUMENT NUMBER: 144:252537
TITLE: Circumventing tolerance at the T cell or the antigen-presenting cell surface: antibodies that ligate CD40 and **OX40** have different effects
AUTHOR(S): Hochweller, Kristin; Sweenie, Claire H.; Anderton, Stephen M.
CORPORATE SOURCE: Institute of Immunology and Infection Research, School of Biological Sciences, University of Edinburgh,

Edinburgh, UK
SOURCE: European Journal of Immunology (2006), 36(2), 389-396
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH Verlag GmbH & Co. KGaA
DOCUMENT TYPE: Journal
LANGUAGE: English
AB An **adjuvant** can be defined as an agent that non-specifically promotes the immune response to an accompanying antigen. Ligation of CD40 on the surface of the antigen-presenting cell leads to upregulation of **OX40** ligand which, in turn, ligates **OX40** on the activated T cell resulting in prolonged T cell proliferation/survival, boosting the immune response. Thus agonistic anti-CD40 and anti-**OX40** might be viewed as "**adjuvant** antibodies" and have been shown in diverse exptl. systems to either boost immune responses or prevent the establishment of immunol. tolerance. Here the authors describe that both these antibodies are able to prevent the induction of tolerance induced using sol. peptide antigen. However, unlike lipopolysaccharide, they are not sufficient to convert tolerance to immunity (i.e. they are not true **adjuvants** in this system). Using mice that are prone to either Th1 or Th2 immunity under identical immunization conditions, the authors show that the effects of anti-**OX40** are quant. - boosting whichever response is dominant. In contrast, anti-CD40 boosts Th1 immunity and converts a Th2 response to Th1. The authors conclude that, although these two antibodies seem to impact on the same mol. pathway of costimulation to prevent tolerance, their effects are qual. distinct and their use cannot be viewed as interchangeable.
OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 20 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Cited References

ACCESSION NUMBER: 2005:1262728 CAPLUS
DOCUMENT NUMBER: 144:5409
TITLE: Eradication of Helicobacter infection by activation of stomach mast cells
INVENTOR(S): Velin, Dominique; Michetti, Pierre
PATENT ASSIGNEE(S): Universite De Lausanne, Switz.
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005113603	A1	20051201	WO 2005-IB1344	20050518

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,

MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: WO 2004-IB1597 A 20040518
US 2004-521543P P 20040519

AB The present invention relates to a novel method and novel compns. for preventing and/or treating a disease caused by or assocd. with Helicobacter in a mammal. According to the invention, a disease caused by or assocd. with Helicobacter in the mammal is prevented and/or treated by administering with preventive and/or therapeutically effective amt. of a compn. capable of activating mast cells in the stomach of the mammal. Furthermore, the invention provides a compn. capable of activating mast cells in the stomach of a mammal,which leads to an increase of the expression of mast cell proteases 1 and/or 2 or related mast cell activation markers as well as a method for eradicating Helicobacter from the stomach of the mammal.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 21 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:423996 CAPLUS
DOCUMENT NUMBER: 143:272144
TITLE: CD134 as target for specific drug delivery to auto-aggressive CD4+ T cells in **adjuvant** arthritis
AUTHOR(S): Boot, Elmieke P. J.; Koning, Gerben A.; Storm, Gert; Wagenaar-Hilbers, Josee P. A.; van Eden, Willem; Everse, Linda A.; Wauben, Marca H. M.
CORPORATE SOURCE: Department of Pharmaceutics, Utrecht Institute for Pharmaceutical Sciences, Utrecht University, Utrecht, Neth.
SOURCE: Arthritis Research & Therapy (2005), 7(3), R604-R615
CODEN: ARTRCV; ISSN: 1478-6362
URL: http://arthritis-research.com/content/pdf/ar1722.pdf
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English

AB T cells have an important role during the development of autoimmune diseases. In **adjuvant** arthritis, a model for rheumatoid arthritis, the authors found that the percentage of CD4+ T cells expressing the activation marker CD134 (**OX40** antigen) was elevated before disease onset. Moreover, these CD134+ T cells showed a specific proliferative response to the disease-assocd. epitope of mycobacterial heat shock protein 60, indicating that this subset contains auto-aggressive T cells. The authors studied the usefulness of CD134 as a mol. target for immune intervention in arthritis by liposomes coated with a CD134-directed monoclonal antibody as a drug targeting system. Injection of anti-CD134 liposomes s.c. in the hind paws of pre-arthritic rats resulted in targeting of the majority of CD4+CD134+ T cells in the popliteal lymph nodes. Furthermore, the authors showed that anti-CD134 liposomes bound to activated T cells were not internalized. However, drug delivery by these liposomes could be established by loading anti-CD134 liposomes with the dipalmitate-derivatized cytostatic agent 5'-fluorodeoxyuridine. These liposomes specifically inhibited the proliferation of activated CD134+ T cells in vitro, and treatment with anti-CD134 liposomes contg. 5'-fluorodeoxyuridine resulted in the amelioration of **adjuvant** arthritis. Thus, CD134 can be used as a marker for auto-aggressive CD4+ T cells early in arthritis, and specific liposomal targeting of drugs to these cells via CD134 can be employed to downregulate disease development.

OS.CITING REF COUNT: 10 THERE ARE 10 CAPLUS RECORDS THAT CITE THIS

RECORD (10 CITINGS)
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 22 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:237942 CAPLUS
DOCUMENT NUMBER: 140:285969
TITLE: TNF Receptor-Associated Factor 5 Limits the Induction of Th2 Immune Responses
AUTHOR(S): So, Takanori; Salek-Ardakani, Shahram; Nakano, Hiroyasu; Ware, Carl F.; Croft, Michael
CORPORATE SOURCE: Division of Molecular Immunology, La Jolla Institute for Allergy and Immunology, San Diego, CA, 92121, USA
SOURCE: Journal of Immunology (2004), 172(7), 4292-4297
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The TNF receptor-assocd. factor (TRAF) family of mols. acts as adapter proteins for signaling pathways initiated by several members of the TNF receptor (TNFR) superfamily. TRAF5-/- animals are viable and have normal development of the immune system despite interacting with several TNFR family members. A clear role for TRAF5 has yet to emerge. **OX40** (CD134) interacts with TRAF5, suggesting that this pathway could be involved in regulating T cell differentiation into Th1 or Th2 cells. In tissue culture, **OX40** stimulation of TRAF5-/- T cells resulted in a pronounced Th2 phenotype with elevated levels of IL-4 and IL-5. Similarly, in vivo immunization with protein in **adjuvant** in the presence of an agonist anti-**OX40** Ab resulted in enhanced Th2 development in TRAF5-/- mice. Addnl., lung inflammation induced by T cells, which is critically controlled by **OX40**, was more pronounced in TRAF5-/- mice, characterized by higher levels of Th2 cytokines. These results suggest that TRAF5 can limit the induction of Th2 responses, and that TRAF5 can play a role in modulating responses driven by **OX40** costimulation.
OS.CITING REF COUNT: 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 23 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:647855 CAPLUS
DOCUMENT NUMBER: 139:228908
TITLE: Cholera Toxin Promotes the Induction of Regulatory T Cells Specific for Bystander Antigens by Modulating Dendritic Cell Activation
AUTHOR(S): Lavelle, Ed C.; McNeela, Edel; Armstrong, Michelle E.; Leavy, Olive; Higgins, Sarah C.; Mills, Kingston H. G.
CORPORATE SOURCE: Department of Biochemistry, Immune Regulation Research Group, Trinity College, Dublin, Ire.
SOURCE: Journal of Immunology (2003), 171(5), 2384-2392
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB It has previously been reported that cholera toxin (CT) is a potent mucosal **adjuvant** that enhances Th2 or mixed Th1/Th2 type responses to

coadministered foreign Ag. Here the authors demonstrate that CT also promotes the generation of regulatory T (Tr) cells against bystander Ag. Parenteral immunization of mice with Ag in the presence of CT induced T cells that secreted high levels of IL-4 and IL-10 and lower levels of IL-5 and IFN-γ. Ag-specific CD4+ T cell lines and clones generated from these mice had cytokine profiles characteristic of Th2 or type 1 Tr cells, and these T cells suppressed IFN-γ prodn. by Th1 cells. Furthermore, adoptive transfer of bone marrow-derived dendritic cells (DC) incubated with Ag and CT induced T cells that secreted IL-4 and IL-10 and low concns. of IL-5. It has previously been shown that IL-10 promotes the differentiation or expansion of type 1 Tr cells. Here the authors found that CT synergized with low doses of LPS to induce IL-10 prodn. by immature DC. CT also enhanced the expression of CD80, CD86, and **OX40** (CD134) on DC and induced the secretion of the chemokine, macrophage inflammatory protein-2 (MIP-2), but inhibited LPS-driven induction of CD40 and ICAM-I expression and prodn. of the inflammatory cytokines/chemokines IL-12, TNF-α, MIP-1α, MIP-1β, and monocyte chemoattractant protein-1. The authors' findings suggest that CT induces maturation of DC, but, by inducing IL-10, inhibiting IL-12, and selectively affecting surface marker expression, suppresses the generation of Th1 cells and promotes the induction of T cells with regulatory activity.

OS.CITING REF COUNT: 81 THERE ARE 81 CAPLUS RECORDS THAT CITE THIS RECORD (81 CITINGS)
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 24 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | CAPLUS References

ACCESSION NUMBER: 2002:657966 CAPLUS
DOCUMENT NUMBER: 137:200248
TITLE: Antigen-pulsed dendritic cells for use as vaccine or vaccine **adjuvant** against Cryptococcus neoformans infection
INVENTOR(S): Thomas, Elaine K.
PATENT ASSIGNEE(S): Immunex Corporation, USA
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002066053	A2	20020829	WO 2001-US48288	20011214
WO 2002066053	A3	20040108		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002246657	A1	20020904	AU 2002-246657	20011214
US 20050048645	A1	20050303	US 2003-451200	20031014

PRIORITY APPLN. INFO.: US 2001-259653P P 20010104
WO 2001-US48288 W 20011214

AB Antigen-expressing, activated dendritic cells are disclosed. Such dendritic cells are used to present Cryptococcus neoformans antigens to T cells, and can be useful in vaccination or treatment protocols. Other cytokines can be used in sep., sequential or simultaneous combination with the activated, antigen-pulsed dendritic cells. Also disclosed are methods for stimulating an immune response using the antigen-expressing, activated dendritic cells.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 25 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2002:657958 CAPLUS
DOCUMENT NUMBER: 137:200247
TITLE: Dendritic cell mobilization agent, dendritic cell maturation agent, apoptosis-causing agent, T cell-enhancing agent and tumor antigen for cancer immunotherapy
INVENTOR(S): Thomas, Elaine K.; Lyman, Stewart D.; Lynch, David H.; De Smedt, Thibaut N.; Maliszewski, Charles R.
PATENT ASSIGNEE(S): Immunex Corporation, USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002066044	A2	20020829	WO 2001-US46254	20011023
WO 2002066044	A3	20040325		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2426659	A1	20020829	CA 2001-2426659	20011023
AU 2001297677	A1	20020904	AU 2001-297677	20011023
AU 2001297677	B2	20070705		
EP 1427813	A2	20040616	EP 2001-273795	20011023
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004529102	T	20040924	JP 2002-565602	20011023
MX 2003003632	A	20030910	MX 2003-3632	20030424
US 20040131587	A1	20040708	US 2003-381160	20030616

PRIORITY APPLN. INFO.: US 2000-242868P P 20001024
WO 2001-US46254 W 20011023

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB An improved method for treatment of a tumor bearing subject comprising administering to said subject a combination of from two to five agents is disclosed. The agents may be agents that mobilize dendritic cells, agents

that cause apoptosis and/or necrosis of tumor cells, chemoattractants, agents that stimulate maturation of dendritic cells, and agents that enhance an anti-tumor response of a T cell.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L2 ANSWER 26 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Info References

ACCESSION NUMBER: 2002:643556 CAPLUS
DOCUMENT NUMBER: 137:336658
TITLE: The role of costimulation and **adjuvants** in the development of T cell effector and memory responses
AUTHOR(S): Maxwell, Joseph Ryan
CORPORATE SOURCE: Oregon State Univ., Corvallis, OR, USA
SOURCE: (2002) 260 pp. Avail.: UMI, Order No. DA3029570 From: Diss. Abstr. Int., B 2002, 62(10), 4454
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable

L2 ANSWER 27 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Info References

ACCESSION NUMBER: 2002:353295 CAPLUS
DOCUMENT NUMBER: 136:368437
TITLE: Agents inducing mobilization, maturation, and activation of dendritic cells and T cell-enhancing factor are used for treating infection
INVENTOR(S): Lynch, David H.; De Smedt, Thibaut N.; Maliszewski, Charles R.; Butz, Eric A.; Miller, Robert E.; Thomas, Elaine K.
PATENT ASSIGNEE(S): Immunex Corporation, USA
SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2002036141	A2	20020510	WO 2001-US44834		20011030
WO 2002036141	A3	20030821			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2002032447	A5	20020515	AU 2002-32447		20011030
US 20040247563	A1	20041209	US 2004-399116		20040527
PRIORITY APPLN. INFO.:			US 2000-245721P	P	20001102
			WO 2001-US44834	W	20011030

AB An improved method for treatment of an individual suffering from or at risk for an infectious disease, comprising administering to said

individual a combination of from two to five agents is disclosed. The agents may be agents (e.g. Flt3L) that mobilize dendritic cells, agents (e.g. TRAIL) that cause death or growth inhibition of infectious agents, chemoattractants, agents (e.g. CD40L) that stimulate maturation of dendritic cells, and agents (e.g. IL-15, **OX40** agonist, 4-1BB agonist) that enhance an immune response of an effector T cell. Antigen-expressing, activated dendritic cells are disclosed. Such dendritic cells are used to present antigens (specifically, antigens derived from infectious agents) to T cells, and can be useful in vaccination protocols. Useful cytokines can be used in sep., sequential or simultaneous combination with the activated, antigen-pulsed dendritic cells. Also disclosed are methods for stimulating an immune response using the antigen-expressing, activated dendritic cells.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 28 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 2002:329037 CAPLUS
DOCUMENT NUMBER: 136:400188
TITLE: Contrasting the roles of costimulation and the natural **adjuvant** lipopolysaccharide during the induction of T cell immunity
AUTHOR(S): Maxwell, Joseph R.; Ruby, Carl; Kerkvliet, Nancy I.; Vella, Anthony T.
CORPORATE SOURCE: Division of Immunology, University of Connecticut Health Center, Farmington, CT, 06030, USA
SOURCE: Journal of Immunology (2002), 168(9), 4372-4381 CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The requirements for circumventing tolerance induction in favor of memory T cell development are poorly understood. Although two signals (Ag and costimulation) are necessary to drive effective T cell clonal expansion, few memory T cells remain after the response wanes. The **adjuvant** LPS can increase nos. of long-lived Ag-specific T cells, but its mechanism of action is not understood. In this report, it is shown that LPS, when combined with two-signal stimulation, profoundly enhances T cell survival in vivo. This survival does not appear to be dependent on the cytokines TNF-α, IL-1β, IL-6, and IFN-γ, nor is it dependent on the transcription factor NF-κB. However, in vivo proliferation of NF-κB-deficient T cells was comparable to that of wild-type T cells, yet their early accumulation in the lymph nodes was severely reduced unless the mice were treated with LPS and an agonistic CD40 mAb. Most importantly, the authors found that activation of two different costimulatory signals, CD40 and **OX40**, could not substitute for LPS in rescuing T cells from peripheral deletion. Perhaps surprisingly, these data show that LPS delivers a qual. different signal than multiple costimulatory signals.

OS.CITING REF COUNT: 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 29 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:280607 CAPLUS
DOCUMENT NUMBER: 136:384521
TITLE: **OX40**: targeted immunotherapy - implications for tempering autoimmunity and enhancing vaccines
AUTHOR(S): Weinberg, Andrew D.
CORPORATE SOURCE: Earle A. Chiles Research Institute, Providence Portland Medical Center, Portland, OR, 97213, USA
SOURCE: Trends in Immunology (2002), 23(2), 102-109
CODEN: TIRMAE; ISSN: 1471-4906
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. **OX40** (CD134), a membrane-bound member of the tumor-necrosis-factor-receptor superfamily, is expressed primarily on activated CD4+ T cells. Recently, several groups have reduced clin. signs of autoimmunity in animal models by blocking the **OX40**-**OX40**-ligand interaction or depleting **OX40**+ T cells. By contrast, engagement of **OX40** in the setting of active immunization has potent **adjuvant** properties, leading to enhanced cytokine prodn. and increased nos. of antigen-specific memory T cells. These potent **adjuvant** effects lead to an enhancement of anti-tumor responses. **OX40** has several unique features that make it a clin. relevant target. They include: (1) T cells isolated from a site of inflammation that express **OX40** are T cells that have been stimulated recently through the T-cell receptor in vivo; (2) **OX40** is only expressed on T cells found at the site of inflammation, therefore, targeting this receptor does not interfere with the peripheral T-cell repertoire; and (3) the biol. function of **OX40** is limited primarily to effector CD4+ T cells, which are a major source of cytokines to induce and maintain ongoing immune responses. **OX40** is expressed on Ag-stimulated CD4 T cells found at the site of inflammation in multiple disease states. Blockade or engagement of **OX40** during inflammation can lead to beneficial outcomes in autoimmunity or the enhancement of vaccine development, resp.
OS.CITING REF COUNT: 69 THERE ARE 69 CAPLUS RECORDS THAT CITE THIS RECORD (69 CITINGS)
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 30 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:892533 CAPLUS
DOCUMENT NUMBER: 136:367960
TITLE: Breaking immunological tolerance through **OX40** (CD134)
AUTHOR(S): Bansal-Pakala, Pratima; Croft, Michael
CORPORATE SOURCE: Division of Immunochemistry, La Jolla Institute for Allergy and Immunology, San Diego, CA, USA
SOURCE: TheScientificWorld [online computer file] (2001), 1, 633-635
CODEN: THESAS; ISSN: 1532-2246
URL: http://216.25.253.202/TSWJaudit/pdf/2001.21.341.pdf
PUBLISHER: TheScientificWorld, Inc.
DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English
AB A review describes a study which demonstrated that T cell tolerance can be prevented and, more significantly, reversed by providing signals through a novel inducible mol. expressed on the surface of T cells, called **OX40**

(CD134). The ability to reverse tolerance through **OX40** indicated that targeting this mol. may have tremendous benefit as an **adjuvant** to antigen-specific therapies aimed at augmenting immune function. The study suggested that it may be feasible to enhance anti-tumor immunity by reversing tolerance of tumor-specific T cells, even after the tumor is well established.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 31 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Cited References

ACCESSION NUMBER: 1997:534123 CAPLUS
DOCUMENT NUMBER: 127:219345
ORIGINAL REFERENCE NO.: 127:42721a,42724a
TITLE: Tumor necrosis factor blockade in actively induced experimental autoimmune encephalomyelitis prevents clinical disease despite activated T cell infiltration to the central nervous system
AUTHOR(S): Korner, Heinrich; Lemckert, Frances A.; Chaudhri, Geeta; Etteldorf, Susanne; Sedgwick, Jonathon D.
CORPORATE SOURCE: Centenary Institute Cancer Medicine Cell Biology, Royal Prince Alfred Hospital, Camperdown, 2050, Australia
SOURCE: European Journal of Immunology (1997), 27(8), 1973-1981
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: Wiley-VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Recently, exptl. autoimmune encephalomyelitis (EAE) in the rat, passively transferred using myelin basic protein (MBP)-reactive encephalitogenic CD4+ T cells, was preventable by administration of a p55-tumor necrosis factor-IgG fusion protein (TNFR-IgG). This was despite quant. and qual. normal movement of these MBP-specific T cells to the central nervous system (CNS). To extend these findings, the effect of TNFR-IgG on EAE actively induced by injection of MBP in complete Freund's **adjuvant** was examd. This form of EAE in the rat typically involves an acute, self-limiting neurol. deficit, substantial CNS inflammation, but minimal demyelination. Administration of TNFR-IgG prior to onset of disease signs completely prevented the neurol. deficit or markedly reduced its severity. This blockade of clin. disease was dissocd. from wt. loss which occurred at the same tempo and magnitude as in control rats exhibiting neurol. signs of disease such as paralysis. The timing of TNF blockade was crit. as established clin. disease was relatively refractory to TNFR-IgG treatment. Activated CD4+ T cells expressing normal or elevated levels of VLA4, major histocompatibility complex class II, MRC **OX40**, and CD25 were isolated from or immunohistochem. localized in the CNS of clin. healthy rats treated before disease onset. There was a redn. of the amt. of other inflammatory leukocytes in the CNS of these treated animals but, more importantly, the activation state of inflammatory leukocytes, as well as that of microglia isolated from treated animals, was reduced. Thus, TNFR-IgG, when administered before disease onset, appears to act by inhibiting an effector function of activated T cells and possibly other inflammatory leukocytes necessary to bring about the neurol. deficit. However, while TNF is a critically important cytokine for the early events leading to initiation of EAE, it is not a necessary factor in the acute neurol. deficit characteristic of this form of EAE, once disease onset has

occurred.
OS.CITING REF COUNT: 57 THERE ARE 57 CAPLUS RECORDS THAT CITE THIS RECORD (57 CITINGS)

L2 ANSWER 32 OF 32 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 1996:86617 CAPLUS
DOCUMENT NUMBER: 124:143409
ORIGINAL REFERENCE NO.: 124:26675a,26678a
TITLE: OX-40 antibody enhances for autoantigen specific Vβ8.2+ T cells within the spinal cord of Lewis Rats with autoimmune encephalomyelitis
AUTHOR(S): Weinberg, A. D.; Lemon, M.; Jones, A. J.; Vainiene, M.; Celnik, B.; Buenafe, A. C.; Culbertson, N.; Bakke, A.; Vandenbark, A. A.; Offner, H.
CORPORATE SOURCE: Veteran Affairs Center, Oregon Health Science University, Portland, OR, USA
SOURCE: Journal of Neuroscience Research (1996), 43(1), 42-9
CODEN: JNREDK; ISSN: 0360-4012
PUBLISHER: Wiley-Liss
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The Vβ8.2 T cell receptor (TCR) component is the predominant Vβ gene product assocd. with antigen specific CD4+ T cell response to the major encephalitogenic epitope of myelin basic protein (MBP) in Lewis rats. Lewis rats were actively immunized with MBP in complete Freund's **adjuvant** and the Vβ8.2 pos. and neg. cells were analyzed for IFN-γ mRNA prodn. and OX-40 cell surface expression during the onset of EAE. The Vβ8.2+ T cells isolated from the spinal cord produced the majority of mRNA for IFN-γ and also showed a marked enhancement for OX-40 expression compared to Vβ8.2+ T cells isolated from the lymph nodes. Only a fraction of IL-2 receptor pos. T cells examd. ex vivo from the inflammatory compartments co-expressed the OX-40 antigen. These results suggested that OX-40 cell surface expression could be used to identify and isolate the most recently activated T cells ex vivo. OX-40+ T cells isolated from the spinal cord were highly enriched for the Vβ8.2 T cell receptor component compared to OX-40- or unsorted spinal cord lymphocytes. OX-40+ T cells isolated from the spinal cord had an enhanced response to MBP, whereas OX-40+ cells isolated from the lymph nodes responded to both MBP and purified protein deriv. These data suggest that activated T cells can be isolated and characterized with the OX-40 antibody which only respond to the antigens present at the local site. The data also imply that isolation of OX-40+ T cells will be useful in identifying Vβ biases and autoantigen specific cells within inflamed tissues even when the antigen specificity is unknown.
OS.CITING REF COUNT: 28 THERE ARE 28 CAPLUS RECORDS THAT CITE THIS RECORD (28 CITINGS)

=>